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REVIEW

Respiratory changes with deep diving

K. Segadal*, A. Gulsvik**, G. Nicolaysen***

Respiratory changes with deep diving. K. Segadal, A. Gulsvik, G. Nicolaysen.
ABSTRACT: Deep diving refers to saturation diving to a depth of more than 180 m (1.9 MPa ambient pressure). In the 1990s diving to 400 m may be necessary on the Norwegian continental shelf. The safety margins are narrow and the respiratory system is subject to great strain at such depths. The respiratory resistance increases and the dynamic lung volumes are reduced as the pressure increases due to enhanced gas density. Helium is used together with oxygen as breathing gas and its lower density partly normalises the dynamic lung volumes. The respiratory system puts clear limitations on intensity and duration of physical work in deep diving. Systematic studies of lung mechanics, gas exchange and respiratory regulation in the different phases of deep dives are lacking. Detection of occupational respiratory disorder following diving are dependent on long-term follow-up.
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* Norwegian Underwater Technology Centre A/S,

** Department of Thoracic Medicine, University of Bergen,

*** Institute of Physiology University of Oslo.

Correspondence: Kåre Segadal, c/o NUTEC, Pb 6, N-5034 YTRE LAKSEVAG, Norway.

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Diving to more than 50 m cannot safely be done with compressed air as a breathing gas. High gas density causes high breathing resistance, and high partial pressures of O_2 and N_2 may cause O_2 poisoning and N_2 narcosis [1]. Commercial off-shore diving was introduced to Europe with the exploitation of oil and gas in the North Sea in the early 1960s. Development of fields in deeper and deeper waters meant that the oil industry needed diving to depths close to 300 m in 1983–1984. At present the Norwegian oil industry needs to master 360 m and in the 1990s diving to 400 m.

There is considerable experience with diving down to around 180 m with He-O_2 mixtures (heliox) as inspired gas. The medical and physiological stresses at these depths are not worse than with air diving to 50 m. The term "deep diving" is now commonly used for going deeper than 180 m.

Such deep dives are now exclusively done as saturation dives, *i.e.* diving where the biological inert gases (*e.g.* H_2, He, Ne or N_2) in all tissues of the body are in equilibrium with the respired gas. Depending on the actual inert gas this equilibrium might take up to 24 h to develop. A saturation dive includes a compression, a bottom and a decompression phase. The diver sleeps, rests and eats in a pressure vessel (living or deck decompression chamber) at the surface on a ship, rig or platform. During the bottom phase he is daily transported down to the seabed by a diving bell (hence the name "bell diving") to work in the water. The bottom phase may last up to 2 weeks and the pressure in both chamber and bell is constantly kept equal to the pressure at the work site (saturation pressure). By compression to more than 180 m the high pressure neurological syndrome (HPNS) may occur. This includes dizziness, tremor, nausea, and changes of consciousness as well as EEG changes. The prevalence of HPNS depends on compression rate and saturation depth. The compression rate is slower with deeper depths (*e.g.* 6 h to 200 m and 48 h to 400 m). To avoid decompression sickness the decompression rate must be less than 30 m a day in heliox saturation diving.

Physical aspects

The increase in hydrostatic pressure is around one atmosphere per 10 m depth of seawater. The unit meter of seawater (msw) is used for over pressure relative to the atmospheric pressure at sea level. It refers to seawater with a specific density of 1.02 so that 10 msw is exactly 100 kPa (1 bar or 750 mm Hg). In the USA pressure is often expressed in normal atmospheres (1 atm=101.3 kPa) absolute (ATA). At 400 msw the over pressure is 4.0 MPa while absolute pressure is 4.1 MPa (40.5 ATA).

When the ambient pressure increases it will be equilibrated to all parts of the body. For solid and liquid tissues this is of minor consequence as they are nearly incompressible. For an ideal gas, pressure and volume are inversely related and the density is proportional to molecular weight and pressure. The density of heliox with a Po_2 of 70 kPa is around 4 $kg \cdot m^{-3}$ at 200 msw and 7 $kg \cdot m^{-3}$ at 400 msw. Air has a density of about 1 $kg \cdot m^{-3}$ at 0 msw and 40 $kg \cdot m^{-3}$ at 400 msw. Since the

partial pressure of a gas is proportional to the molecular fraction, a heliox of 1% exhibits a Po_2 of approximately 40 kPa at 400 msw. Heliox deviates insignificantly from an ideal gas up to pressures around 400 msw [2].

Molecular diffusivity is inversely proportional to the square root of the molecular weight. The diffusivity for He is therefore 2.6 times higher than for N_2. The diffusivity decreases with increasing pressure and density. The diffusivity for O_2 in air is reduced by 80% when the pressure increases from 0 to 40 msw. The diffusivity for 1% O_2 in heliox at around 150 msw is the same as for O_2 in air at 40 msw [3].

The flow resistance with turbulent flow is proportional to velocity and density but independent of the viscosity of the gas. In laminar flow the resistance is proportional to viscosity and independent of density and velocity. Under normal conditions and rest there will be laminar flow in all airways except the largest where there may be partial turbulence. Only with a ventilation of about 60 $l \cdot min^{-1}$, or more, will the turbulent pressure losses dominate in airways with a diameter of more than 4 mm [4]. With increasing pressure the viscosity is unchanged while the density, and therefore the degree of turbulence, increases. During resting ventilation at 400 MSW the flow is turbulent in all large airways.

In a maximal expiratory manoeuver the flow will be limited by three different mechanisms, each dominating at different lung volumes. The capacity of the respiratory muscles to generate force is the limiting factor for maximal flow when exhaling the first approximate 25% of the vital capacity (VC). In the remaining part of the manoeuver effort independent flow limitation operates [5]. Maximal expiratory flow in the middle 50% of the VC is limited by the speed of wave propagation by the airway walls, being inversely related to the square root of the gas density [6]. When exhaling the last 25% of the VC, viscous pressure losses may cause the pressure within some small airways to fall so much below the surrounding pleural pressure that there will be partial collapse of the airway. Further increase of pleural pressure will give more collapse and no increase of flow. The maximal expiratory flow will then be highly dependent on viscosity and less on the density of the gas since the flow is laminar.

The resistance of the breathing apparatus will also increase by density and pressure. This is minimized by introducing servomechanisms for opening and closing the breathing valve. The breathing equipment normally gives an additional dead space of 200–400 ml.

By immersion in water the central blood volume, cardiac output and diuresis are increased [7]. With zero flow the gas pressure will be the same in all airways and alveoli, from the mouth to basal parts of the lung. The surrounding pressure from the water on the body will, however, increase from the upper to the lower parts of the body because of the hydrostatic pressure increase. The hydrostatic imbalance for the lungs is the difference between the gas pressure in the airways at zero flow and the surrounding pressure. Standing erect with water to the neck, the hydrostatic pressure will give around 3 kPa overpressure on the chest wall. Functional residual capacity (FRC) is reduced to about 50% and expiratory reserve volume (ERV) to around 25% of normal [8]. The airways are compressed: the thoracic wall and lungs must work in a position where they are stiffer; therefore both the flow resistance and elastic work of breathing are increased [9, 10]. Hydrostatic imbalance and increased gas density lead to dyspnoea and decrease the work capacity [11]. The imbalance may be abolished and the dyspnoea decreased by positive pressure breathing which normalises ERV and FRC [12, 13].

The composition of breathing gas during deep diving is based on physiological and practical considerations. The important physical parameters of the gas mixture are density, viscosity, diffusivity, sound velocity, heat capacity and heat conductivity. The two latter factors are important for the diver's thermal balance. Respiratory heat loss increases as the heat capacity rises with increasing pressure. The ambient water temperature during deep diving in the North Sea is only 2–5°C so the breathing gas must be heated [14]. Sound velocity is crucial to voice production; *e.g.* divers get a "Donald Duck" voice in heliox at high pressure.

Gas components are divided into: inert gases, respiratory gases (O_2 and CO_2) and contaminates. He, H_2 and Ne, alone or in mixtures, may replace N_2 as inert gas as they have little narcotic potential and low density and He is the obvious choice at depths from 50 to 400 msw. Addition of 5–10% N_2 to heliox ("N_2-trimix") has been implemented to reduce the HPNS symptoms [15]. H_2-trimix with around 50% H_2 seems equally as effective for avoiding HPNS symptoms as N_2-trimix and will, in addition, give less airway resistance than ordinary heliox [16]. The main problem with H_2 mixtures is the explosion hazard, but the risk of decompression sickness may also be greater because H_2 has higher fat solubility [17].

To secure adequate arterial oxygenation the Po_2 is kept at a higher level than normal (20 kPa), usually around 50 kPa and seldom below 35 kPa or above 100 kPa. The risk of O_2 poisoning of lungs and central nervous system depends on partial pressure and duration of exposure.

Pulmonary O_2 poisoning is characterized by a perivascular and interstitial inflammatory reaction with oedema and capillary endothelial damage [18]. The first symptoms are substernal soarness and pain, especially with deep inhalation. VC, compliance and CO transfer factor (T_{LCO}) may be reduced [19].

Contaminants in the inspiration gas may be caused by improper purification during production but may also be created by special work processes, especially hyperbaric welding (*e.g.* ozone) and by endogen production from the diver (*e.g.* acetaldehyde and CO). Degassing from equipment (*e.g.* solvents) is a bigger problem in a closed system and at increased pressure than under normal conditions. Recirculation of breathing gas is necessary to keep the inert gas consumption within acceptable limits, but this may cause accumulation of toxic substances.

The breathing gas for diving is produced and stored without humidity to avoid corrosion and icing, but such gas will cause drying of the airways. Breathing equipment that humidifies the diving gas has been developed and is currently under test.

Methods

Technical problems with sensors and signal transmissions make it difficult to measure respiratory variables during deep diving. Simulated deep diving is done in pressure chambers with an internal gas pressure similar to the hydrostatic pressure at the required sea depth. Wet and dry simulated dives are separate entities. In dry diving the chambers are only filled with gas; in simulated wet diving one chamber is partly filled with water to allow submersion.

Even in a dry chamber there are methodological problems; the sensors must mechanically withstand the pressure and the frequency response may change. Gas samples and electrical signals must be fed through the chamber wall *via* pressure safe penetrators. The observer and the subject are separated and the communication problems are significant because of voice distortion.

In wet simulated diving there are further problems as the sensors must be protected against water intrusion. The measuring devices may impinge on both the diver's movements and the function of his life support equipment, thereby disturbing the work situation that is to be investigated.

Deep divers

There is a common labour market for divers in the North Sea irrespective of whether they are in the British or Norwegian sector. Out of some 2000 divers certified in Norway to go beyond 50 msw (bell diving) about 300 are Norwegian and only half of these may be on active duty. Most deep divers are men of 25 years or older; few continue such diving after the age of 40. The Norwegian Directorate of Public Health [20] and the British Health and Safety Executive [21] have laid down stringent conditions as to health and physical fitness before licensing a bell diver. No special regulations exist for deep dives beyond 180 msw; it is up to the individual oil or diving company to make any additional fitness demands and usually they require more sophisticated tests of the central nervous system and lungs. Even small deviations from normal lung function, usually not considered of any importance, may result in disqualification of a candidate. Reduced lung compliance may increase the susceptibility to barotrauma [22]. Measured maximal O_2 uptake on a treadmill is often required and we recommend measurement of CO_2 sensitivity and TLco.

Acute changes

Acute changes are those appearing during the compression, bottom, or decompression phase of a deep dive.

Dynamic lung volumes and forced ventilatory flow rates decrease with increasing gas density. The reduction is approximately proportional to the gas density raised to a power (x) characteristic for each of the indices. In the part of expiration where speed of wave transmission is a limiting factor, the power is highest (x=approximately 0.5). Thus, maximal air flow at 75% VC (FEF_{75}) is approximately inversely proportional to the square root of the gas density. In the late phase of a VC expiration (*e.g.* FEF_{15}) the maximal expiratory flow is almost independent of gas density. For intermediate volumes the power exponent is between 0.5 and 0. These effects of density have been described both in models [23] and experimentally during dives to 500 MSW and with gas densities up to 16 $kg \cdot m^{-3}$ [24–28]. This implies that if FEF_{50} were 5.5 $l \cdot s^{-1}$ at surface it would be 2.5 $l \cdot s^{-1}$ at 400 msw when heliox is used as breathing gas.

At lung volumes where maximal expiratory flow is force dependent, any increased airway resistance may be counteracted through increased force due to decreased contraction velocity of the expiratory muscles. A four times higher transpulmonary pressure has been observed at 490 MSW than at the surface [27] during forced expiration. Maximal peak expiratory flow (PEF) is consequently better conserved than FEF_{50} during deep dives [27–29].

Maximal voluntary ventilation (MVV) varies with gas density to about the same extent as PEF [27–29]. Breathing at higher lung volumes where a larger expiratory force can be generated and the airway resistance is less [27] probably explains why the reduction in MVV with increased gas density is not more prominent. The airway resistance increases with increasing gas density [30] and the respiratory muscles must exert greater mechanical work in order to maintain unchanged minute ventilation. The increase in frictional respiratory work is partly counteracted by breathing at a higher lung volume and with larger tidal volume than at the surface [27, 28, 31–33]. These latter adaptations, however, increase the elastic work, the net effect still being a reduced total respiratory work relative to that expected purely from the increase in gas density.

In connection with compression the dynamic indices decrease more than expected from the increase in gas density. A quick recovery and then stabilization is observed, however, when a constant depth is maintained [24, 27, 28, 34]. The explanation for these transient changes is not known. A vagal stimulation in connection with compression may induce transient airway constriction and thus be part of the explanation [24]. The secondary improvement may be due to an adaptation to high density breathing gas through training of respiratory muscles [35].

Static lung volumes show, as expected, only small changes in connection with deep dives. Thus, lung compliance (elastic behaviour) appears to remain unchanged with dives to 500 msw as long as O_2 toxicity is avoided [26, 27, 36]. A reduction in VC of up to 12% has been observed when compression was very quick [24], while during the bottom phase it is normalized or may even increase above the surface value [28, 34].

The gas exchange is primarily limited through the reduced ability to ventilate resulting from increased gas density. A reduced gas exchange could also be due to a less effective gas mixing in the lungs or increased physiological dead space. This dead space has been shown to enlarge with increasing gas density in both dogs and

man. At 650 MSW it is about twice the surface volume [33]. An unchanged or even reduced alveolo-arterial O_2 difference has been observed at high gas density [37, 38]. A better ventilation perfusion ratio could explain such a finding, but in dogs it has been reported [39] that this ratio is less ideal when air, in contrast to heliox, is used as breathing gas even if the latter gas has a lower density. In a recent review [40] on gas mixing in the lungs this apparent paradox is used as an example of how incomplete our present knowledge is of gas mixing and gas exchange in the lungs.

The physical work capacity is less during experimental wet deep dives [13, 27, 41] than during dry dives [28, 33, 42]. Dry dives to 300 msw [28] and 400 msw [42] are accompanied by a reduction of 13 and 30%, respectively, of the maximal work capacity obtained at the surface. The divers experience a marked dyspnoea and the workloads are tolerated for only a few minutes. During dives to 650 msw [33] and workloads implying O_2 uptake exceeding 2 $l \cdot min^{-1}$ the alveolar ventilation was only 50% of that at the surface. The arterial Pco_2 increased to about 7 kPa.

During experimental wet dives to 430–550 msw, the divers could only perform physical work for a few minutes due to intense dyspnoea [13, 27, 41]. The breathing equipment and thus the additional breathing resistance may explain part of the reduction in performance. Hydrostatic imbalance could not explain the observations since the achievement was not improved when the imbalance was neutralized [13, 41].

What kind of practical work does the deep diver perform? During a simulated dive to 478 msw (thermoneutral water) four divers without diving suits mounted and demounted well-head components. At the surface this work was completed in 10 min with a $\dot{V}o_2$ of 2.0 $l \cdot min^{-1}$. This same work was executed just as quickly at depth [43]. Heart rate recordings from shallow air dives (1–20 msw) indicate that the $\dot{V}o_2$ required to perform typical work tasks will never exceed 2 $l \cdot min^{-1}$ [44]. During a simulated dive to 500 msw at the Norwegian Underwater Technology Centre (NUTEC) six divers performed similar work under more realistic conditions; the water was cold (6–8°C) and hot water diving suits were worn [42].

During dives to 350 msw [45] and later also to 450 msw and 360 msw (unpublished observations) measurements of respiration were taken during work periods of 3.5 h. For most of these work periods the $\dot{V}o_2$ was about 1 $l \cdot min^{-1}$, but during shorter periods with hard work it was 2 $l \cdot min^{-1}$. Sub-maximal work was performed as effectively as by diving to 5 msw, but some divers complained of extreme fatigue.

No objective measurements are available of working intensity during deep diving at sea. Performances which demand considerable manual work (repair of pipes, installation of platforms, attempts to salvage valves) have been done at between 300 and 330 msw. Automatic well-heads have been installed by divers at 307 msw outside Brazil [46]. French deep divers recently stayed in the water for 4 h at 530 msw while breathing H_2-trimix [47]. They carried out simple underwater tasks without great problems.

Regulation of respiration

The respiratory system will, if it is exposed to increased ventilation resistance counteract a reduction in ventilation in three ways: 1) Passive mechanical circumstances increase the ability for the respiratory muscles to generate force, *i.e.* by changed rate of contractions and length of muscle; 2) mechanical sensors in the bronchi, lung and chest wall (tension, length, flow and pressure receptors) record increased resistance and induce activity of the respiratory muscle; 3) chemo-receptors may, *via* the respiratory centre, give increased stimulation [48]. The first mechanism may act immediately, the second after one breath, while the third mechanism is slower. The compensation is very good in awake situations and the ventilation is maintained during very high resistance against breathing. The respiratory frequency is reduced and the tidal volume increased. This may partly be due to conscious reactions against discomfort *via* the brain to the respiratory centre [49]. The muscles in the larynx may also compensate by reducing the airway resistance [50]. However, the mechanisms of compensation are poorer and slower during sleep [51, 52].

The ventilatory response to increased CO_2 is mostly reduced when exposed to increased breathing resistance. When awake the mechanical compensation mechanisms diminish the reduction in ventilation [53] which may be due to increased efficiency of the diaphragm or recruitment of other inspiratory muscles [54].

Compensation mechanisms also appear in deep divers with increased airway resistance. In 14 deep divers at 360 msw and heliox atmosphere the ventilatory CO_2 response was almost unchanged immediately after compression. Later during the dive it was reduced to 60% of pre-dive values which indicates an adaptation [55]. In four divers at 300 msw the ventilatory response was reduced by 20% [28]. The compensations are not so good when diving with air. The ventilatory response is reduced by 50% when diving to 50 msw [56, 57]. Extreme high gas density (21 $kg \cdot m^{-3}$ with Ne mixtures) at 360 msw [58] caused a ventilatory response of only 15% of values at the surface in two subjects. The differences in results may be due to the fact that compression with heliox to high pressure may change the neuromuscular coupling and the sensitivity of receptors and respiratory centre. Inert gas narcosis may also reduce the compensatory mechanism. Some studies of ventilatory CO_2 response to loading indicate that the compensation is greater in individuals with a low unloaded response [59–61]: however the opposite has also been reported [62].

Response to hypoxia may be of less importance since the inspiratory gases are hyperoxic. If, on the other hand, the diver is accidently exposed to a hypoxic gas mixture then a normal response to hypoxia is of the utmost importance. Sensitivity to hypoxia has not yet been systematically examined during deep diving.

Sub-acute changes

Static and dynamic lung volumes after a deep dive may be somewhat different from pre-dive values. VC is mostly unchanged while the residual volume (RV) may increase by 15% [63]. The total lung capacity increased by 5% in 24 divers who dived to 360 msw or deeper [63]. These changes normalized after 1 month [63]. The inspiratory and expiratory peak flow may be reduced by 10% [24], but several authors have recorded less [28, 64] or no change in dynamic lung volume [63]. Opposite effects may act on static and dynamic lung volumes. Training of the respiratory muscles due to increased airway resistance, toxic effects of hyperoxic and polluted gas mixtures and effects due to compression and decompression in itself are possible explanations for these small differences.

TLCO has been found to be lowered by 10 to 15% of pre-dive values in several studies after deep diving. TLCO mostly normalize within 1 month [63, 65, 66] but not in all studies [64, 67]. This reduction in transfer factor may be due to micro-emboli of gas bubbles or thrombi in the lung, damage to the alveolo-capillary membrane or alveolar oedema [68]. If such changes in gas exchange are present during decompression they may impede the removal of gas bubbles in the lung. Maximal Vo_2 after a deep dive (300–450 msw) is reduced by 7–16% [28, 66, 67]. The change in working capacity seems to be dependent on duration and depth of the dive [69]. Normalization of working capacity seems to occur within 1 month [66, 67]. It is possible that pulmonary hypertension, which has been documented in animal experiments after decompression [68] is the common denominator for both reduction in maximal Vo_2 and transfer factor [67]. An increased ventilatory response to CO_2 is observed in several divers immediately after deep diving [55, 70, 71]. The causes are not known, but it is reasonable to suspect changes in the central nervous system or on receptor level. Similar changes have not been found for the hypoxia response [72].

Chronic changes

Many professional divers have, during their career, done different forms of diving, *i.e.* sports diving, helmet diving, navy diving, "normal" bell diving and deep diving. Later damage may be due to compression/decompression profiles, hyperoxia, pollutants, different activities, equipment faults and accidents. Further factors which complicate detection of chronic effects from deep diving are self selection for diving, reluctance of men to be followed up on account of the possibility of losing the job, and the confounding effects of, for example, smoking, alcohol and working environments not related to diving. To detect pulmonary changes it is necessary to have long-term follow-ups of at least 5–10 years duration with a considerable number of subjects. This investigation must include measurement of lung mechanics, gas exchange and respiratory regulation. The exposure of the divers is crudely estimated from log books as regards maximal depth, type and duration of dives.

Dynamic and static lung volumes have been examined in a great number of English divers. It has been suggested that VC in young divers is on average 20% greater than for individuals in a reference sample: training has been claimed to be of some importance as the VC is positively correlated with the maximal diving depth [73, 74]. However, a large VC may be a selection factor for the occupation of diving [75]. Nevertheless, the fall in VC with age is more than anticipated [76]. Forced expiratory volume in one second (FEV_1) is also somewhat greater in young divers than predicted but not so great as the VC; FEV% is thus often lower than expected. FEV_1 is not correlated to previous maximal diving depth but there is evidence that it falls faster after the age of 30 years than expected [76]. Reductions in maximal flows at low lung volumes have been observed [73] and are positively correlated to number of years as a diver [74]. A morphometric study [77] of divers' lungs shows a correlation between FVC and increased mean cord length. The authors suggest that distension of the alveoli may cause narrowing of small airways.

These changes from follow-ups of VC and dynamic lung volumes have been found in divers who have not been down deeper than 180 msw. Longitudinal studies of divers who regularly dive deeper are not available. Studies of gas exchange after several deep dives are not reported, but such studies are of great importance due to the observed sub-acute reduction of TLCO.

Ventilatory response to CO_2 is in some air divers only half that measured in healthy men [78, 79]. Whether this is due to selection or to the influence of diving is unknown. This observation is not confirmed in deep divers [55].

Conclusions

Divers have increased airways resistance and increased workload for breathing due to high gas density. Gas exchange in the lung is changed, the working capacity is reduced, and some divers experience extreme breathlessness and fatigue.

Some of the problems observed in deep diving may be solved by new developments in breathing equipment, training and the use of hydrogen in the inspiratory gas. The respiratory function must be examined before and after all new diving procedures to exclude respiratory damage due to diving. It is still unknown whether the observed sub-acute changes in the respiratory system after deep diving normalize completely. If diving is repeated at short intervals the effects may accumulate and cause persistent changes.

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Modifications respiratoires au cours de la plongée en profondeur. K. Segadal, A. Gulsvik, G. Nicoloysen.

RÉSUMÉ: La plongée en profondeur se rapporte à la plongée en saturation à une profondeur de plus de 180 mètres (pression ambiante: 1.9 MPa). Au cours des années 1990, la plongée jusqu'à 400 mètres pourrait être nécessaire sur le plateau continental norvégien. Les marges de sécurité sont étroites et le système respiratoire est sujet à des efforts considérables à de telles profondeurs. La résistance respiratoire augmente et les volumes pulmonaires dynamiques sont diminués au fur et à mesure de l'augmentation de pression due à une densité accrue des gaz. L'hélium est utilisé avec l'oxygène comme gaz respiratoire, et sa densité plus faible normalise partiellement les volumes pulmonaires dynamiques. Le système respiratoire impose des limitations évidentes à l'intensité et à la durée de l'effort physique lors de la plongée en profondeur. Des études systématiques de la mécanique pulmonaire, des échanges gazeux et de la régulation respiratoire aux différentes phases de la plongée en profondeur, font défaut. La détection de maladies respiratoires professionnelles liées à la plongée dépendra du follow-up à long terme.
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